groups. Higher NT-proANP levels were found in group II compared to group IV (0.1425 vs. 0.078 nmol/L, p=0.04) and higher BNP levels in group II compared to group III (897.37 vs. 419.4 pg/ml, p=0.016). One day before profound cardiogenic shock occurred NT-pro ANP, BNP and big ET-1 were elevated in group II compared to group III (0.126 vs. 0.094 nmol/L, p=0.049; 848.23 vs. 342.2 pg/ml, p=0.016; 20.7 vs. 5.65 pg/ml, p=0.019 resp.). NT-proBNP remained unchanged during treatment in groups II and III but decreased significantly in group IV (p = 0.9, p = 0.6 and p = 0.025 resp.).
Conclusion: While routine parameters did not predict the clinical course, natriuretic peptides showed significant differences between groups on admission and before clinical deterioration.
Daily measurement of natriuretic peptides may be used to determine optimal time-point for HTx or VAD implantation in patients with severe end-stage heart failure.

1049-108 B-Type Natriuretic Peptide and Chest Radiograph Findings as Indicators of Systolic Dysfunction in Patients Presenting With Acute Dyspnea

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Background: The diagnosis of left ventricular (LV) systolic dysfunction may be difficult in acute dyspneic patients. Chest radiographs are routinely performed, but are not always diagnostically definitive. Objective: To compare the diagnostic value of chest radiograph findings with that of B-type natriuretic peptide (BNP) testing as indicators of LV ejection fraction < 35%. Methods: The Breathing Not Properly Study was a multinational study evaluating the diagnostic accuracy of BNP in 1586 patients presenting with acute dyspnea. Chest radiographs and echocardiograms permitting estimation of LV ejection fraction were available in 709 of the patients. BNP was measured on arrival in all patients. Two cardiologists blinded to BNP results reviewed all clinical data and categorized patients as to whether they had acute heart failure (63.8%) or not (36.2%). Results: Diagnostic information from chest radiographs and from BNP is presented in the Table. Multivariate logistic regression showed that both BNP ≥ 100 pg/ml (OR: 8.1 (95% CI: 3.8-17.6)) and chest radiograph findings of cardiomegaly (OR: 2.3 (95% CI: 1.4-3.8)) and pleural effusion (OR: 1.8 (95% CI: 1.2-2.9)) added significant, predictive information above historical and clinical predictors of systolic dysfunction. Conclusion: In patients presenting with acute dyspnea, BNP and chest radiographs provide complementary diagnostic information. Both methods should both be utilized in the early evaluation of LV systolic dysfunction.

Diagnostic performance of chest radiograph variables and BNP

Variable	Sensitivity (%)	Specificity (%)	Accuracy (%)
Cardiomegaly	85	50	61
Cephalisation	34	80	66
Pleural effusion	31	84	67
BNP ≥ 100 pg/mL	94	41	57

1049-109 Evaluation of B-Type Natriuretic Peptide Levels in Normal and Pre-Eclamptic Women During Pregnancy

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OBJECTIVE: B-type natriuretic peptide (BNP) is synthesized in cardiac ventricular tissue in response to volume expansion and pressure overload. It is an approved marker for the diagnosis of congestive heart failure (CHF) in patients with dyspnea in an acute care setting. BNP levels correlate to wedge pressure, severity of CHF, and medical prognosis. BNP has also been used to screen for ventricular dysfunction in high-risk patients such as diabetics and hypertensives. The objective of this study was to determine whether BNP levels fluctuated during pregnancy, and if BNP could be a useful diagnostic tool in preeclamptic women.
DESIGN: We studied 195 BNP levels in 78 women longitudinally from the first trimester to term. An additional 33 patients admitted with preeclampsia (9 mild, 24 severe) were studied and compared to 21 normal controls with term pregnancies. Plasma BNP values were determined using a standard point of care assay.
RESULTS: BNP levels did not change significantly across gestation in normal pregnant women. In early pregnancy, BNP values are 25 pg/ml ± 2.4, and at term, values are 22 pg/ml ± 3.6. BNP remains stable at this level throughout the midtrimester. Patients with mild preeclampsia had higher BNP levels than controls, however this difference was not statistically significant (44.4 v. 22.1 pg/ml, p >.05). Women with severe preeclampsia, in the absence of evidence of clinical cardiac disease, had mean BNP levels of 124.6 v. 22.1pg/ml in controls (p=0.017).
CONCLUSION: In normal pregnancies, BNP values are stable throughout gestation. Patients with severe preeclampsia have elevated BNP values. This may reflect ventricular stress and/or subclinical cardiac dysfunction associated with the disease process.

1049-110 Circulating Beta-Atrial Natriuretic Peptide in Human Coronary Artery Disease: A New Marker for Stage A Heart Failure?

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Background: Studies have reported the presence of an antiparallel dimer form of atrial natriuretic peptide (ANP) named β-ANP in both atrial tissue and plasma of normal humans. β-ANP has been shown to be elevated in patients with severe congestive heart failure (CHF). Its biological significance is that it may have reduced cGMP enhancing actions compared to native ANP and its formation may be secondary to states of oxidative stress within the myocardium and plasma. To date, it is unknown whether β-ANP is elevated in patients with CAD independently of co-presence of left ventricular dysfunction (LVD) which represents Stage A of CHF. The aim of this study was to determine the concentration of β-ANP in patients with CAD. We hypothesized that plasma concentration of β-ANP is elevated in patients with CAD independently of concomitant LVD.
Methods: We prospectively enrolled 58 subjects (33 men), average age 66 (41 to 84), with CAD referred to the Mayo Clinic Cardiac Catheterization Laboratory. Ejection fraction (EF) was assessed by echocardiogram. Subjects were divided in two subgroups according to EF (EF> or EF< 50%). Twenty normal subjects served as a control group. β-ANP was measured by RIA. * indicates p<0.05 versus CAD.
Results: β-ANP was elevated in CAD patients compared to normal subjects (25.4±4.2 CAD versus 7±0.7 normal*). When analyzed by subgroups, β-ANP tended to increase in the subgroup (n=13) with EF<50% compared with subjects with EF ≥ 50% (n=45) (p=0.05). Importantly, β-ANP was significantly elevated in the subgroup with EF ≥ 50% as compared to normal (22.4±4.4 CAD ≥ 50% EF versus 7±0.7 normal*). An inverse correlation was observed between β-ANP and EF (Spearman r= -0.3219; 95% C.I. -0.539 to -0.064; p=0.0065).
Conclusion: We report for the first time that β-ANP is elevated in patients with CAD and is inversely correlated with EF. More importantly, β-ANP is elevated in subjects with CAD and normal EF (Stage A CHF). β-ANP may be a useful marker to identify patients with CAD independently of co-existing LVD who are at risk for progression to CHF increasing our available biomarkers for cardiovascular disease.

1049-111 Plasma B-Type Natriuretic Peptide in Obstructive Sleep Apnea

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Background: Obstructive sleep apnea (OSA) has been associated with several cardiovascular diseases. Brain natriuretic peptide (BNP), released by the ventricles in response to high wall tension, has been linked to development of hypertension, heart failure and nocturnal angina. OSA is also associated with these disease conditions and activates those mechanisms implicated in BNP release. The aim of the present study was to clarify whether plasma BNP is acutely or chronically increased by OSA, either in otherwise healthy subjects, or in subjects diagnosed with both OSA and chronic heart failure (CHF), or OSA and other cardiovascular diseases.
Methods: Plasma BNP levels were measured in 23 patients with moderate to severe OSA who were either otherwise healthy (n=10) or who also had concomitant cardiovascular disease (n=13, 5 patients with both OSA and CHF, 8 patients with OSA and non-CHF cardiovascular disease). BNP was obtained on three occasions: first before sleep, then after 5 hours of untreated OSA, and again in the morning, after 4 hours of effective treatment with continuous positive airway pressure (CPAP). BNP levels were also measured in 10 closely matched, normal healthy subjects at similar time points.
Results: Baseline BNP levels were similar in the 10 otherwise healthy OSA subjects and the 10 normal controls before sleep (6.1±10.2 vs. 6.2±10.2 pg/mL respectively, *P*=0.96) and were unaffected by several hours of untreated OSA and by acute CPAP treatment. BNP levels at baseline were higher, compared to controls, in 5 OSA patients with CHF (90.2±14.4 pg/mL, *P*=0.0016) and in 8 OSA patients with other non-CHF cardiovascular diseases (31.7±11.4 pg/mL, *P*=0.05). These high levels of BNP remained stable through the night in the OSA with CHF and the OSA with non-CHF cardiovascular disease groups, completely unaffected either by several hours of acute untreated OSA or by CPAP treatment.
Conclusions: Despite the metabolic and mechanical stresses elicited by OSA, OSA in and of itself does not increase plasma BNP in otherwise healthy subjects during wakefulness. OSA does not elicit acute sleep-related changes in BNP in healthy OSA subjects nor in patients with coexisting cardiovascular disease, including CHF.

1049-112 Utility of B-Type Natriuretic Peptide and ProBNP in Evaluation of Patients Receiving Natrecor Therapy (UBET Study)

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BACKGROUND: B type natriuretic peptide (BNP) is synthesized in cardiac ventricles as a prohormone (108 amino acids) and during the release process is cleaved into the active hormone BNP (AA 77-108) and an inactive fragment proBNP (AA 1-76). We determined the effect of Natrecor (human recombinant BNP) infusions on the concentrations of BNP and proBNP.
METHODS: Three groups of acutely decompensated congestive heart failure (CHF) patients received Natrecor (2 ug/kg iv bolus followed by a 0.01 ug/kg/min infusion) for 24, 36 or 48 hours (N = 10, 9 and 8 respectively). Serial blood samples were collected during and after the infusion, BNP (Biosite and Bayer Diagnostics) and proBNP (proBrain Natriuretic Peptide, Roche Diagnostics) were measured.